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      (FILE 'HOME' ENTERED AT 08:44:44 ON 08 JUN 2005)

      FILE 'REGISTRY' ENTERED AT 08:52:23 ON 08 JUN 2005
L1             1 S BUPROPION/CN

      FILE 'CAPLUS, USPATFULL' ENTERED AT 08:52:56 ON 08 JUN 2005
L2          2242 S L1 OR BUPROPION
L3          9371 S (TREAT? OR PREVENT? OR CONTROL? OR INHIBIT?) (3A) (RLS OR RES
L4            68 S (TREAT? OR PREVENT? OR CONTROL? OR INHIBIT?) (3A) (LEG? (2A)
L5            68 S (TREAT? OR PREVENT? OR CONTROL? OR INHIBIT?) (3A) (LEG? (2A)
L6          9436 S L3 OR L4
L7            11 S L6 (P) L2

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protein kinase (e.g. RWJ67657) and phosphoinositide 3-kinase. Retinoids may be one of the few potential treatments capable of reversing alveolar destruction in COPD, and a number of compds. are in clin. trial (e.g. all-trans-retinoic acid). Talniflumate (MSI-1995), an inhibitor of human calcium-activated chloride channels, has been developed to treat mucous hypersecretion. In addition, the purinoceptor P2Y2 receptor agonist diquafosol (INS365) is undergoing clin. trials to increase mucus clearance. The challenge to transferral of these new compds. from preclin. research to disease management is the design of effective clin. trials. The current scarcity of well characterized surrogate markers predicts that long-term studies in large nos. of patients will be needed to monitor changes in disease progression.

REFERENCE COUNT: 205 THERE ARE 205 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review. Chronic obstructive pulmonary disease (COPD) is a common, smoking-related, severe respiratory condition characterized by progressive, irreversible airflow limitation. Current **treatment** of COPD is **symptomatic**, with no drugs capable of halting the relentless progression of airflow obstruction. Better understanding of the airway inflammation, oxidative stress and alveolar destruction that characterize COPD has delineated new disease targets, with consequent identification of novel compds. with therapeutic potential. These new drugs include aids to smoking cessation (e.g. **bupropion**) and improvements to existing therapies, for example long-acting rather than short-acting bronchodilators, as well as combination therapy. New antiproteases include acyl-enzyme and transition state inhibitors of neutrophil elastase (e.g. sivelestat and ONO-6818), matrix metalloprotease inhibitors (e.g. batimastat), cathepsin inhibitors and peptide protease inhibitors (e.g. DX-890 [EPI-HNE-4] and trappin-2). New antioxidants include superoxide dismutase mimetics (e.g. AEOL-10113) and spin trap compds. (e.g. N-tert-butyl-(α-phenylnitrone)). New anti-inflammatory interventions include phosphodiesterase-4 inhibitors (e.g. cilomilast), inhibitors of tumor necrosis factor-α (e.g. humanised monoclonal antibodies), adenosine A2a receptor agonists (e.g. CGS-21680), adhesion mol. inhibitors (e.g. bimosiamose [TBC1269]), inhibitors of nuclear factor-.vkappa.B (e.g. the naturally occurring compds. hypoestoxide and (-)-epigallocatechin-3-gallate) and activators of histone deacetylase (e.g. theophylline). There are also selective inhibitors of specific extracellular mediators such as chemokines (e.g. CXCR2 and CCR2 antagonists) and leukotriene B4 (e.g. SB201146), and of intracellular signal transduction mols. such as p38 mitogen activated protein kinase (e.g. RWJ67657) and phosphoinositide 3-kinase. Retinoids may be one of the few potential treatments capable of reversing alveolar destruction in COPD, and a number of compds. are in clin. trial (e.g. all-trans-retinoic acid). Talniflumate (MSI-1995), an inhibitor of human calcium-activated chloride channels, has been developed to treat mucous hypersecretion. In addition, the purinoceptor P2Y2 receptor agonist diquafosol (INS365) is undergoing clin. trials to increase mucus clearance. The challenge to transferral of these new compds. from preclin. research to disease management is the design of effective clin. trials. The current scarcity of well characterized surrogate markers predicts that long-term studies in large nos. of patients will be needed to monitor changes in disease progression.

L7 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2000:885378 CAPLUS

DOCUMENT NUMBER: 135:86980

TITLE: Bupropion SR reduces periodic limb movements associated with arousals from sleep in depressed patients with periodic limb movement disorder

AUTHOR(S): Nofzinger, Eric A.; Fasiczka, Amy; Berman, Susan; Thase, Michael E.

CORPORATE SOURCE: Department of Psychiatry, University of Pittsburgh

School of Medicine, Pittsburgh, PA, USA
Journal of Clinical Psychiatry (2000), 61(11), 858-862
CODEN: JCLPDE; ISSN: 0160-6689
PUBLISHER: Physicians Postgraduate Press, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background: Antidepressant-induced periodic limb movement disorder (PLMD) may limit the tolerability of some antidepressant medications and interfere with treatment response. Given the role of dopamine in PLMD and the effects of **bupropion** sustained-release (SR) on central dopaminergic function, we hypothesized that **bupropion** SR would not be associated with antidepressant-induced PLMD. Method: In an expanded case-series design, we compared the effects of **bupropion** SR, after about 10 wk of **treatment**, on measures of **PLMD**, depression, and sleep in 5 depressed (Research Diagnostic Criteria) patients who also met criteria for having pretreatment PLMD. Depression was measured using the Beck Depression Inventory and the Hamilton Rating Scale for Depression. Patients were considered to have PLMD if polysomnog. recordings showed > 5 periodic limb movements/h of sleep that were associated with arousals from sleep. Results: **Bupropion** SR treatment was associated with a reduction in measures of PLMD and an improvement in depression. Conclusion: These results show that **bupropion** SR is not associated with antidepressant-induced PLMD. Rather, **bupropion** SR treatment reduces objective measures of PLMD in depressed patients with the disorder.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Background: Antidepressant-induced periodic limb movement disorder (PLMD) may limit the tolerability of some antidepressant medications and interfere with treatment response. Given the role of dopamine in PLMD and the effects of **bupropion** sustained-release (SR) on central dopaminergic function, we hypothesized that **bupropion** SR would not be associated with antidepressant-induced PLMD. Method: In an expanded case-series design, we compared the effects of **bupropion** SR, after about 10 wk of **treatment**, on measures of **PLMD**, depression, and sleep in 5 depressed (Research Diagnostic Criteria) patients who also met criteria for having pretreatment PLMD. Depression was measured using the Beck Depression Inventory and the Hamilton Rating Scale for Depression. Patients were considered to have PLMD if polysomnog. recordings showed > 5 periodic limb movements/h of sleep that were associated with arousals from sleep. Results: **Bupropion** SR treatment was associated with a reduction in measures of PLMD and an improvement in depression. Conclusion: These results show that **bupropion** SR is not associated with antidepressant-induced PLMD. Rather, **bupropion** SR treatment reduces objective measures of PLMD in depressed patients with the disorder.

L7 ANSWER 5 OF 11 USPATFULL on STN
ACCESSION NUMBER: 2004:151042 USPATFULL
TITLE: Use of **bupropion** for **treating** **restless legs** syndrome
INVENTOR(S): Robertson, David W., Glenview, IL, UNITED STATES
Krafft, Grant, Glenview, IL, UNITED STATES LR

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004115263	A1	20040617	
APPLICATION INFO.:	US 2003-645649	A1	20030820	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-405943P	20020826 (60)